Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSSPTA1743MXC

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	SEP 09	ACD predicted properties enhanced in REGISTRY/ZREGISTRY
NEWS	4	OCT 03	MATHDI removed from STN
NEWS	5	OCT 04	CA/CAplus-Canadian Intellectual Property Office (CIPO) added to core patent offices
NEWS	6	OCT 13	New CAS Information Use Policies Effective October 17, 2005
NEWS	7	OCT 17	STN(R) AnaVist(TM), Version 1.01, allows the export/download of CAplus documents for use in third-party analysis and visualization tools
NEWS	8	OCT 27	Free KWIC format extended in full-text databases
NEWS	9	OCT 27	DIOGENES content streamlined
NEWS	10	OCT 27	EPFULL enhanced with additional content
NEWS	11	NOV 14	CA/CAplus - Expanded coverage of German academic research
NEWS	12	NOV 30	REGISTRY/ZREGISTRY on STN(R) enhanced with experimental spectral property data
NEWS	13	DEC 05	CASREACT(R) - Over 10 million reactions available

NEWS EXPRESS DECEMBER 02 CURRENT VERSION FOR WINDOWS IS V8.01,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 02 DECEMBER 2005.
V8.0 USERS CAN OBTAIN THE UPGRADE TO V8.01 AT
http://download.cas.org/express/v8.0-Discover/

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 11:27:30 ON 05 DEC 2005

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'STNGUIDE' ENTERED AT 11:27:35 ON 05 DEC 2005

FILE CONTAINS CURRENT INFORMATION.

```
LAST RELOADED: Dec  2, 2005 (20051202/UP).

=> FIL HOME
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.06       0.27

FILE 'HOME' ENTERED AT 11:27:41 ON 05 DEC 2005

=> file biosis
COST IN U.S. DOLLARS                                  SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.21       0.48

FILE 'BIOSIS' ENTERED AT 11:27:49 ON 05 DEC 2005
Copyright (c) 2005 The Thomson Corporation

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 1 December 2005 (20051201/ED)


=> s (rem and sleep)
          7284 REM
           414 REMS
          7515 REM
                 (REM OR REMS)
         59961 SLEEP
            91 SLEEPS
         59985 SLEEP
                 (SLEEP OR SLEEPS)
L1        6890 (REM AND SLEEP)

=> s l1 and (blood or serum or sera)
       2541806 BLOOD
           598 BLOODS
       2541882 BLOOD
                 (BLOOD OR BLOODS)
        571469 SERUM
           627 SERUMS
         84086 SERA
            12 SERAS
        617792 SERUM
                 (SERUM OR SERUMS OR SERA OR SERAS)
         84086 SERA
            12 SERAS
         84091 SERA
                 (SERA OR SERAS)
L2         613 L1 AND (BLOOD OR SERUM OR SERA)

=> s (diagnos? or prognos? or determin?) (3w) l1
PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'ETERMIN?) (3W) L1'
       1141805 DIAGNOS?
        160575 PROGNOS?
       1524763 DETERMIN?
L3        1806 (DIAGNOS? OR PROGNOS? OR DETERMIN?) (3W) L1

=> s l3 and (blood or serum or sera)
       2541806 BLOOD
           598 BLOODS
       2541882 BLOOD
                 (BLOOD OR BLOODS)
        571469 SERUM
           627 SERUMS
         84086 SERA
            12 SERAS
        617792 SERUM
```

```
                (SERUM OR SERUMS OR SERA OR SERAS)
         84086 SERA
            12 SERAS
         84091 SERA
                (SERA OR SERAS)
L4         183 L3 AND (BLOOD OR SERUM OR SERA)

=> s l4 and protein
       1515860 PROTEIN
        583567 PROTEINS
       1743160 PROTEIN
                (PROTEIN OR PROTEINS)
L5           4 L4 AND PROTEIN

=> d l5 1-4 kwic

L5   ANSWER 1 OF 4  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation  on STN
TI   Inflammatory markers and sleep disturbance in major depression.
AB   Objective: This study was conducted to determine whether immune
     activation Occurs in major depression, and to evaluate the associations
     between disordered sleep and markers of inflammation in patients
     with major depressive disorder.  Methods: All-night polysomnography was
     obtained in patients with acute Diagnostic and Statistical
     Manual of Mental Disorders, 4th edition major depressive disorder (n = 22)
     and age-, gender-, and body weight-matched comparison controls (n = 18).
     After the onset of sleep, nocturnal serum levels of
     interleukin-6 (IL-6), soluble intercellular adhesion molecule (sICAM),
     monocyte chemotactic protein (MCP-1), and IL-6 Soluble receptor
     (IL-6sR) were sampled.  Results: As compared with matched controls,
     depressed patients showed significant (p < .05) nocturnal elevations of
     circulating levels of IL-6 and sICAM.  Both sleep latency and
     rapid eye movement (REM) density had moderate correlations with
     IL-6 and sICAM (r's gtoreq 0.30).  Backward regression analyses indicated
     that sleep latency (beta = 0.34, p < .05) and REM
     density (beta = 0.27 p = .09) were better predictors of IL-6 than
     depressive Status.  Similarly, sleep latency (beta = 0.27, p =
     .06) and REM density (beta = 0.32, p = .02) were also better
     predictors of sICAM.  Conclusion: These findings support the hypothesis
     that sleep disturbance is associated with elevated levels of the
     inflammatory markers IL-6 and sICAM.  This relationship was not accounted
     for by.  .  .  findings suggest that the elevations in inflammatory
     markers found in depressive Subjects may be partially the result of
     disturbances of sleep initiation found in this Population.
IT   .  .  .
        Medicine, Medical Sciences); Neurology (Human Medicine, Medical
        Sciences); Psychiatry (Human Medicine, Medical Sciences)
IT   Parts, Structures, & Systems of Organisms
           serum: blood and lymphatics
IT   Diseases
        major depression: behavioral and mental disorders, pathology
        Depression (MeSH)
IT   Diseases
           sleep disturbance: behavioral and mental disorders, nervous
        system disease, symptom
           Sleep Disorders (MeSH)
IT   Chemicals & Biochemicals
        IL-6 soluble receptor [IL-6sR]; inflammatory markers; interleukin-6
        [IL-6]; monocyte chemotactic protein-1 [MCP-1]; soluble
        intercellular adhesion molecule [sICAM]
IT   Methods & Equipment
           Diagnostic and Statistical Manual of Mental Disorders:
        clinical techniques, diagnostic techniques; polysomnography:
        clinical techniques, diagnostic techniques
IT   Miscellaneous Descriptors
        rapid eye movement

L5   ANSWER 2 OF 4  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation  on STN
AB.  .  .  the other is hematopoietic PGDS (H-PGDS) in mast cells and Th2
     lymphocytes.  L-PGDS is the same as beta-trace, a major protein
     in human cerebrospinal fluid, and is also secreted into the seminal plasma
```

and plasma. The L-PGDS concentration in various body. . . and coronary atherosclerosis. H-PGDS is a cytosolic enzyme and is a member of the Sigma class of glutathione S-transferase. We **determined** the X-ray crystallographic structures of H-PGDS and L-PGDS. We also generated the gene-knockout (KO) mice and the human enzyme-overexpressing transgenic mice for each PGDS. L-PGDS-KO mice lacked PGE2-induced tactile allodynia and rebound of non-rapid eye movement **sleep** after **sleep** deprivation. Human L-PGDS-overexpressing transgenic mice showed an increase in non-rapid eye movement **sleep** due to accumulation of PGD2 in the brain after tail clipping. H-PGDS-KO mice showed an allergic reaction weaker than that. . .

IT Major Concepts
Behavior; **Blood** and Lymphatics (Transport and Circulation); Cardiovascular System (Transport and Circulation); Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry. . . (Neural Coordination); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis)

IT Parts, Structures, & Systems of Organisms
Th2 lymphocyte: **blood** and lymphatics, immune system; cerebrospinal fluid: nervous system; mast cell: immune system; plasma: **blood** and lymphatics; seminal plasma: reproductive system

IT Diseases
allergy: immune system disease, etiology
Hypersensitivity (MeSH)

IT Diseases
coronary atherosclerosis: heart disease,. . .

IT Methods & Equipment
x-ray crystallography: crystallographic techniques, laboratory techniques

IT Miscellaneous Descriptors
hematopoiesis; non-**REM sleep**

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Inhibition of tumor necrosis factor in the brain suppresses rabbit **sleep.**

AB Tumor necrosis factor (TNF) is a cytokine that possesses many biological activities, including enhancement of non-rapid-eye-movement **sleep** (NREMS). The role of endogenous TNF in the regulation of spontaneous **sleep** is unknown. If TNF is involved in **sleep** regulation, then reduction of endogenous TNF should suppress spontaneous **sleep.** A soluble TNF-binding **protein** I (TNF-BP I) and a synthetic fragment of TNF-BP I, TNF-R-(159-178), that contains the biologically active region of TNF-BP I, were used. These substances bind TNF and possess TNF-inhibitory activity; their effects on rabbit **sleep** after intracerebroventricular injection were **determined** across a 6-h recording period. Two doses of TNF-BP I (0.05 mu-g and 0.5 mu-g) were administered; the higher dose. . . mu-g) were used. The 25 mu-g and 50 mu-g doses significantly suppressed NREMS. The highest dose (50 mu-g) also decreased **REM sleep.** These results are consistent with the hypothesis that endogenous brain TNF is involved in the regulation of normal **sleep.**

IT Major Concepts
Behavior; Biochemistry and Molecular Biophysics; **Blood** and Lymphatics (Transport and Circulation); Endocrine System (Chemical Coordination and Homeostasis); Nervous System (Neural Coordination); Sense Organs (Sensory Reception)

IT Miscellaneous Descriptors
RAPID EYE MOVEMENT **SLEEP**; TUMOR NECROSIS FACTOR-BINDING **PROTEIN**-1

L5 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

AB. . . a muscular habitus, and muscle stiffness and myokymia were found in all muscles of the extremities. Her stiffness persisted during **sleep.** Her calf muscles were large and a contracture was noticed in ankle joints. There was no evidence of wasting and. . . of the CPK linked IgA was lamda type. All other laboratory tests were normal for the following: urinalysis, ESR, a **blood** count, liver function, kidney function, glucose, rheumatoid factor, CRP, thyroid function, parathyroid function, **serum** electrolytes, ECG, EEG, cranial CT, without slight elevation of IgA, and CSF **protein.** In needle EMG

and surface EMG spontaneous discharges were recorded at rest. These discharges consist of normal motor unit potentials,. . . needle EMG, myotonic discharge was not observed. Nerve conduction velocities were within normal ranges. According to these data, she was **diagnosed** as having Issacs' syndrome (continuous muscle fiber activity syndrome). Carbamazepine, 200 mg daily was administrated and showed a dramatic reversal. . . to carbamazepine. Polysomnography was examined before and after treatment. Before treatment, suppression of muscle fiber discharges was not recognized during **REM sleep**. After treatment, suppression of muscle fiber discharges appeared and % stage was elevated (% stage IV, 3.6% → 11.4%). It was suspected that continuous muscle fiber discharges throughout the **sleep** had an influence on the depth of the **sleep** stage. There have been reports of some 40 cases of syndrome of continuous muscle fiber activity. A number of studies. . .

=> d l5 1-3 ibib, iabs

L5 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:208458 BIOSIS
DOCUMENT NUMBER: PREV200500212330
TITLE: Inflammatory markers and **sleep** disturbance in major depression.
AUTHOR(S): Motivala, Sarosh J. [Reprint Author]; Sarfatti, Avishay; Olmos, Luis; Irwin, Michael R.
CORPORATE SOURCE: Inst NeuropsychiatCousins Ctr Psychoneuroimmunol, Univ Calif Los Angeles, 300 Med Plaza,Suite 3160A, Los Angeles, CA, 90095, USA
smotivala@mednet.ucla.edu
SOURCE: Psychosomatic Medicine, (March 2005) Vol. 67, No. 2, pp. 187-194. print.
ISSN: 0033-3174 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 1 Jun 2005
Last Updated on STN: 1 Jun 2005
ABSTRACT:
Objective: This study was conducted to **determine** whether immune activation Occurs in major depression, and to evaluate the associations between disordered **sleep** and markers of inflammation in patients with major depressive disorder.
Methods: All-night polysomnography was obtained in patients with acute ***Diagnostic*** and Statistical Manual of Mental Disorders, 4th edition major depressive disorder (n = 22) and age-, gender-, and body weight-matched comparison controls (n = 18).
After the onset of **sleep**, nocturnal **serum** levels of interleukin-6 (IL-6), soluble intercellular adhesion molecule (sICAM), monocyte chemotactic **protein** (MCP-1), and IL-6 Soluble receptor (IL-6sR) were sampled.
Results: As compared with matched controls, depressed patients showed significant ($p < .05$) nocturnal elevations of circulating levels of IL-6 and sICAM.
Both **sleep** latency and rapid eye movement (**REM**) density had moderate correlations with IL-6 and sICAM (r's gtoreq 0.30).
Backward regression analyses indicated that **sleep** latency (beta = 0.34, $p < .05$) and **REM** density (beta = 0.27 $p = .09$) were better predictors of IL-6 than depressive Status.
Similarly, **sleep** latency (beta = 0.27, $p = .06$) and **REM** density (beta = 0.32, $p = .02$) were also better predictors of sICAM.
Conclusion: These findings support the hypothesis that **sleep** disturbance is associated with elevated levels of the inflammatory markers IL-6 and sICAM.
This relationship was not accounted for by other confounding factors such as age and body weight.
These findings suggest that the elevations in inflammatory markers found in depressive Subjects may be partially the result of disturbances of ***sleep*** initiation found in this Population.

L5 ANSWER 2 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2004:140670 BIOSIS
DOCUMENT NUMBER: PREV200400135658
TITLE: Functional analyses of lipocalin-type and hematopoietic prostaglandin D synthases.
AUTHOR(S): Urade, Yoshihiro [Reprint Author]; Eguchi, Naomi [Reprint Author]; Aritake, Kosuke [Reprint Author]; Hayaishi, Osamu [Reprint Author]
CORPORATE SOURCE: Department of Molecular Behavioral Biology, Osaka Bioscience Institute, 6-2-4 Furuedai, Suita, Osaka, 565-0874, Japan
SOURCE: Folia Pharmacologica Japonica, (January 2004) Vol. 123, No. 1, pp. 5-13. print.
ISSN: 0015-5691 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: Japanese
ENTRY DATE: Entered STN: 10 Mar 2004
Last Updated on STN: 10 Mar 2004
ABSTRACT:
Prostaglandin (PG) D synthase (PGDS) catalyzes the isomerization of PGH2 to PGD2, which acts as an endogeous somnogen and an allergic mediator.
There are two distinct types of PGDS: one is lipocalin-type PGDS (L-PGDS) localized in the central nervous system, male genitals, and heart; and the other is hematopoietic PGDS (H-PGDS) in mast cells and Th2 lymphocytes.
L-PGDS is the same as beta-trace, a major **protein** in human cerebrospinal fluid, and is also secreted into the seminal plasma and plasma.
The L-PGDS concentration in various body fluids is useful as a marker for various diseases such as renal failure and coronary atherosclerosis.
H-PGDS is a cytosolic enzyme and is a member of the Sigma class of glutathione S-transferase.
We **determined** the X-ray crystallographic structures of H-PGDS and L-PGDS.
We also generated the gene-knockout (KO) mice and the human enzyme-overexpressing transgenic mice for each PGDS.
L-PGDS-KO mice lacked PGE2-induced tactile allodynia and rebound of non-rapid eye movement **sleep** after **sleep** deprivation.
Human L-PGDS-overexpressing transgenic mice showed an increase in non-rapid eye movement **sleep** due to accumulation of PGD2 in the brain after tail clipping.
H-PGDS-KO mice showed an allergic reaction weaker than that of the wild-type mice.

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:80564 BIOSIS
DOCUMENT NUMBER: PREV199698652699
TITLE: Inhibition of tumor necrosis factor in the brain suppresses rabbit **sleep.**
AUTHOR(S): Takahashi, Satoshi; Tooley, Dawn D.; Kapas, Levente; Fang, Jidong; Seyer, Jerome M.; Krueger, James M. [Reprint author]
CORPORATE SOURCE: Dep. Physiol. Biophysics, Univ. Tennessee, Memphis, TN 38163, USA
SOURCE: Pfluegers Archiv European Journal of Physiology, (1995) Vol. 431, No. 2, pp. 155-160.
CODEN: PFLABK. ISSN: 0031-6768.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Feb 1996
Last Updated on STN: 27 Feb 1996
ABSTRACT:
Tumor necrosis factor (TNF) is a cytokine that possesses many biological activities, including enhancement of non-rapid-eye-movement **sleep** (NREMS).
The role of endogenous TNF in the regulation of spontaneous **sleep** is unknown.
If TNF is involved in **sleep** regulation, then reduction of endogenous TNF should suppress spontaneous **sleep.**
A soluble TNF-binding **protein** I (TNF-BP I) and a synthetic fragment of TNF-BP I, TNF-R-(159-178), that contains the biologically active region of TNF-BP I, were used.
These substances bind TNF and possess TNF-inhibitory activity; their effects on

rabbit **sleep** after intracerebroventricular injection were
determined across a 6-h recording period.
Two doses of TNF-BP I (0.05 mu-g and 0.5 mu-g) were administered; the higher dose of TNF-BP I significantly decreased NREMS.
Four doses of TNF-R-(159-178) (0.25 mu-g, 2.5 mu-g, 25 mu-g and 50 mu-g) were used.
The 25 mu-g and 50 mu-g doses significantly suppressed NREMS.
The highest dose (50 mu-g) also decreased **REM sleep.**
These results are consistent with the hypothesis that endogenous brain TNF is involved in the regulation of normal **sleep.**

```
=> s l4 and electrophoresis
        201413 ELECTROPHORESIS
L6           0 L4 AND ELECTROPHORESIS

=> s l4 and marker
        172141 MARKER
        145983 MARKERS
        280535 MARKER
                  (MARKER OR MARKERS)
L7           8 L4 AND MARKER

=> d l7 1-8 kwic
```

L7 ANSWER 1 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Inflammatory **markers** and **sleep** disturbance in major depression.

AB Objective: This study was conducted to **determine** whether immune activation Occurs in major depression, and to evaluate the associations between disordered **sleep** and **markers** of inflammation in patients with major depressive disorder. Methods: All-night polysomnography was obtained in patients with acute **Diagnostic** and Statistical Manual of Mental Disorders, 4th edition major depressive disorder (n = 22) and age-, gender-, and body weight-matched comparison controls (n = 18). After the onset of **sleep**, nocturnal **serum** levels of interleukin-6 (IL-6), soluble intercellular adhesion molecule (sICAM), monocyte chemotactic protein (MCP-1), and IL-6 Soluble receptor (IL-6sR) were sampled.. . . with matched controls, depressed patients showed significant ($p < .05$) nocturnal elevations of circulating levels of IL-6 and sICAM. Both **sleep** latency and rapid eye movement (**REM**) density had moderate correlations with IL-6 and sICAM (r's gtoreq 0.30). Backward regression analyses indicated that **sleep** latency (beta = 0.34, $p < .05$) and **REM** density (beta = 0.27 $p = .09$) were better predictors of IL-6 than depressive Status. Similarly, **sleep** latency (beta = 0.27, $p = .06$) and **REM** density (beta = 0.32, $p = .02$) were also better predictors of sICAM. Conclusion: These findings support the hypothesis that **sleep** disturbance is associated with elevated levels of the inflammatory **markers** IL-6 and sICAM. This relationship was not accounted for by other confounding factors such as age and body weight. These findings suggest that the elevations in inflammatory **markers** found in depressive Subjects may be partially the result of disturbances of **sleep** initiation found in this Population.

IT . . .
Medicine, Medical Sciences); Neurology (Human Medicine, Medical Sciences); Psychiatry (Human Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms
serum: **blood** and lymphatics

IT Diseases
major depression: behavioral and mental disorders, pathology
Depression (MeSH)

IT Diseases
sleep disturbance: behavioral and mental disorders, nervous system disease, symptom
Sleep Disorders (MeSH)

IT Chemicals & Biochemicals
IL-6 soluble receptor [IL-6sR]; inflammatory **markers**; interleukin-6 [IL-6]; monocyte chemotactic protein-1 [MCP-1]; soluble intercellular adhesion molecule [sICAM]

IT Methods & Equipment
Diagnostic and Statistical Manual of Mental Disorders: clinical techniques, **diagnostic** techniques; polysomnography: clinical techniques, **diagnostic** techniques
IT Miscellaneous Descriptors
rapid eye movement

L7 ANSWER 2 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI The association between interleukin-6, **sleep**, and demographic characteristics.
AB We examined the relationship between the pro-inflammatory cytokine IL-6 and **sleep** architecture in 10 healthy men and women. **Blood** was drawn in the early morning for assessment of IL-6 followed by nocturnal **sleep** monitoring with polysominoamphy. **Sleep** records were scored for **sleep** stages using standard criteria. Morning IL-6 levels were positively correlated with **REM** latency after **sleep** onset (p =31, p =.01), percent (%) stage I **sleep** (p =.23 p =.053) % wake after **sleep** onset (WASO) (p =.29, p <.05). IL-6 levels were negatively correlated with **sleep** efficiency (p = -36 p < 01) and slow wave **sleep** (SWS) (p = -.26. p <.05) After controlling for demographic variables including race. gender, age. and BMI multiple hierarchical regression analyses revealed that morning IL-6 levels accounted for a significant portion of the variance of **REM** latency (p <.01 **sleep** efficiency (p <.01), and % WASO (p =.01). IL-6 was no Ionger associated with % stage I **sleep.** SWS, and total **sleep** time after controlling for the demographic characteristics. These findings suggest that the inflammatory **marker** IL-6 is associated with **sleep** quality and that certain individual characteristics such as race, gender, and age modify that relationship. Higher IL-6 levels were associated with lower quality of **sleep** among healthy asymptomatic men and women. Copyright 2004 Elsevier Inc. All rights reserved.
IT . . .
Biochemistry and Molecular Biophysics; Clinical Immunology (Human Medicine, Medical Sciences); Epidemiology (Population Studies)
IT Parts, Structures, & Systems of Organisms
blood: **blood** and lymphatics
IT Chemicals & Biochemicals
interleukin-6: pro-inflammatory cytokine
IT Methods & Equipment
polysomnography: clinical techniques, **diagnostic** techniques
IT Miscellaneous Descriptors
demographic characteristic; **sleep** pattern

L7 ANSWER 3 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
AB. . . is also secreted into the seminal plasma and plasma. The L-PGDS concentration in various body fluids is useful as a **marker** for various diseases such as renal failure and coronary atherosclerosis. H-PGDS is a cytosolic enzyme and is a member of the Sigma class of glutathione S-transferase. We **determined** the X-ray crystallographic structures of H-PGDS and L-PGDS. We also generated the gene-knockout (KO) mice and the human enzyme-overexpressing transgenic mice for each PGDS. L-PGDS-KO mice lacked PGE2-induced tactile allodynia and rebound of non-rapid eye movement **sleep** after **sleep** deprivation. Human L-PGDS-overexpressing transgenic mice showed an increase in non-rapid eye movement **sleep** due to accumulation of PGD2 in the brain after tail clipping. H-PGDS-KO mice showed an allergic reaction weaker than that. . .
IT Major Concepts
Behavior; **Blood** and Lymphatics (Transport and Circulation); Cardiovascular System (Transport and Circulation); Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Enzymology (Biochemistry. . . (Neural Coordination); Reproductive System (Reproduction); Urinary System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
Th2 lymphocyte: **blood** and lymphatics, immune system; cerebrospinal fluid: nervous system; mast cell: immune system; plasma: **blood** and lymphatics; seminal plasma: reproductive system
IT Diseases

allergy: immune system disease, etiology
Hypersensitivity (MeSH)
IT Diseases
coronary atherosclerosis: heart disease,. . .
failure: urologic disease
Kidney Failure (MeSH)
IT Chemicals & Biochemicals
glutathione S-transferase [EC 2.5.1.18]; lipocalin; prostaglandin D synthase [EC 5.3.99.2]: disease **marker**, hematopoietic, lipocalin-type; prostaglandin H-2
IT Methods & Equipment
x-ray crystallography: crystallographic techniques, laboratory techniques
IT Miscellaneous Descriptors
hematopoiesis; non-**REM sleep**

L7 ANSWER 4 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Arterial stiffness increases during obstructive **sleep** apneas.
AB Study Objectives: Obstructive **sleep** apnea (OSA) appears to be an independent risk factor for diurnal systemic hypertension, but the specific biologic **markers** for this association have not been well established. Increased arterial stiffness is an important measure of increased left ventricular load. . . However, arterial stiffness has not been measured in association with obstructive apneas in patients with OSA, nor related to systemic **blood** pressure (BP) activity in this setting. Our objective was to test the hypothesis that arterial stiffness may be utilized as. . . (2) such increased stiffness may occur in the absence of acute BP increase. Design: Prospective, cross-sectional. Setting: A tertiary-care university-based **sleep** and ventilatory disorders center. Patients: Forty-four normo- and hypertensive adult patients (11 women, 33 men) with polysomnographically **diagnosed** moderate to severe OSA. Interventions: N/A. Measurements and Results: Beat-to-beat BP was recorded from the radial artery by applanation tonometry. . . first 15 cardiac cycles following apnea termination ("post apnea"). Mean AAI (+-SD) for the group was significantly increased during NREM **sleep** from early apnea to late apnea (12.02+-2.70% vs 13.35+-3.54%, p<0.05, ANOVA). During **REM** (analyzed in 20 patients), AAI again significantly increased from early apnea to late apnea (11.75+-2.81% vs 13.43+-4.97%). Conversely, neither mean. . . arterial BP was significantly changed from early apnea to late apnea in NREM (SBP 130+-14 mmHg vs 129+-14 mmHg) or **REM** (SBP 128+-22 mmHg vs 127+-21 mmHg). Conclusions: Arterial stiffness increases acutely during obstructive apneas in both NREM and **REM sleep**, in the absence of measurable BP change. These data suggest that arterial stiffness may be a sensitive measure of acute. . .
IT . . .
Sciences)
IT Parts, Structures, & Systems of Organisms
artery: circulatory system
IT Diseases
hypertension: vascular disease
Hypertension (MeSH)
IT Diseases
obstructive **sleep** apnea: respiratory system disease, complications
Sleep Apnea, Obstructive (MeSH)
IT Methods & Equipment
arterial augmentation index: clinical techniques, **diagnostic** techniques; polysomnography: clinical techniques, **diagnostic** techniques
IT Miscellaneous Descriptors
arterial stiffness: modulation; systolic BP [systolic **blood** pressure]: modulation

L7 ANSWER 5 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Light-dark difference in arterial pressure variability during **REM** **sleep** in the rat.
AB We have observed mean arterial pressure (MAP) variability during rapid eye movement (**REM**) **sleep** and brain temperature (Tb) in the

rat during both light and dark periods over 24 h. MAP was measured using a telemetric device with a computer data capture and analysis system. As **markers** of MAP variability, the maximum and coefficient of variation (CV%) of MAP during **REM sleep** were **determined.** The following results were obtained: (a) there was a light-dark difference in MAP during non-**REM** (NREM) **sleep** and Tb during both NREM and **REM sleep**; (b) the increase of MAP in going from NREM to **REM sleep** in the light period was greater than that in the dark period, whereas the increase of Tb in the light period was not different from that in the dark period; (c) the maximum and CV% for MAP during **REM sleep** in the light period were greater than those in the dark period; (d) there was a negative correlation between the average Tb and MAP CV% during **REM sleep.** We suggest that phasic fluctuation of MAP during **REM sleep** may be influenced, in part, by a factor independent of **sleep** mechanisms. Key Words: Arterial pressure-Brain temperature-Rapid eye movement **sleep** -Rat-Circadian rhythm.

IT Major Concepts
Behavior; Biochemistry and Molecular Biophysics; Biosynchronization; **Blood** and Lymphatics (Transport and Circulation); Cardiovascular System (Transport and Circulation); Cell Biology; Metabolism; Nervous System (Neural Coordination); Physiology; Radiation Biology;. . .

IT Miscellaneous Descriptors
BRAIN TEMPERATURE; CHRONOBIOLOGY; RAPID EYE MOVEMENT **SLEEP**

L7 ANSWER 6 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI Genetic influences on EEG **sleep** and the human circadian clock: A twin study.

AB The study of neuroendocrine and **sleep** abnormalities in major depressive disorders has been the focus of major interest in the past few years. However, while **sleep** and neuroendocrine research in neuropsychiatric disorders has progressed considerably during the last few years, conceptional and methodological advances in **sleep** and neuroendocrine physiology are still needed for further understanding of the basic aspects of **sleep** and to clarify the control and significance of the temporal fluctuations of the neuroendocrine systems. In particular, identification of the genetic mechanisms governing **sleep** regulation are of interest. In this respect, twin studies constitute a powerful method for identifying genetic influences on human physiological variables. In a first study, we explored the **sleep** patterns of 26 pairs of noncohabiting normal male twins (both mono- and dizygotic). The results indicate that a significant genetic effect is found for some **sleep** variables. Stages 2, 4, and delta **sleep** as well as waking are substantially **determined** by genetic factors, in contrast to stage **REM** which seems to be mainly affected by nongenetic influences. These data thus provide consistent evidence that some aspects of human **sleep** are genetically **determined.** In a second study we analyzed the 24-hour profile of plasma cortisol in 21 pairs of male twins. The 24-hour profile of plasma cortisol is the most widely used **marker** of the human circadian clock: Its study offers the possibility of assessing the status of the human circadian clock and of **determining** whether genetic factors affect human circadian rhythmicity. In the protocol, **blood** was sampled every 15 min and circadian rhythmicity was characterized by measures of amplitude, phase, and overall waveshape. A genetic. . .

IT Major Concepts
Behavior; Biosynchronization; **Blood** and Lymphatics (Transport and Circulation); Clinical Chemistry (Allied Medical Sciences); Endocrine System (Chemical Coordination and Homeostasis); Genetics; Metabolism; Nervous System. . .

IT Miscellaneous Descriptors
CORTISOL; DELTA **SLEEP**; ELECTROENCEPHALOGRAPHY; NOCTURNAL NADIR; PLASMA LEVEL; PULSATILE TEMPORAL VARIABILITY; RAPID EYE MOVEMENT; REGULATION; STAGE 2; STAGE 4; WAKING

L7 ANSWER 7 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI USEFULNESS OF **SLEEP** AND NEUROENDOCRINE TESTS AS BIOLOGICAL

MARKERS OF DEPRESSION IN CHILDREN AND ADOLESCENTS.

AB In this article, we systematically reviewed the results of application of biological **markers** of depression to children and adolescents. Concerning **sleep** EEG, only three studies on a total of twelve among 267 depressed children and adolescents aged 6 to 19 years found the typical **sleep** abnormalities described in depressed adults (eg, shortened **REM** latency and decreased **sleep** efficiency). Most authors insisted on the age-related **sleep** changes as a major confounding factor. Two studies of the effect of antidepressant therapy on **sleep** showed a decrease in **sleep** efficiency but a discrepancy in the evolution of **REM** latency. Concerning the dexamethasone suppression test, twenty studies including 374 depressed children and adolescents (3-20 years) and 533 psychiatric controls. . . be considered as interesting, despite the lack of agreement among authors on various methodological parameters (dose of dexamethasone, times of **blood** sampling, method of cortisol assay...) and the composition of control groups which often comprise subjects presenting disorders very close to. . . in two studies, showed limited interest. In contrast, the study of growth hormone secretion, performed in one centre, could present **diagnostical** usefulness. In conclusion, biological **markers** of depression in children and adolescents should still be considered as research tools and be part of a multidisciplinary approach. . .

L7 ANSWER 8 OF 8 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

TI RED **BLOOD** CELL-PLASMA CHOLINE RATIO IN ELDERLY DEPRESSED AND DEMENTED PATIENTS.

AB In further study of red **blood** cell (RBC) and plasma choline concentrations in 160 elderly subjects, we found no significant differences in RBC/plasma choline ratios among. . . RBC/plasma choline ratios > 1.9. Thus, it now appears that static RBC choline levels cannot be recommended as a specific **marker** of Alzheimer's dementia. However, within subgroups of these **diagnostic** categories, **determined** by RBC/plasma choline ratios ≤ 1.9 or > 1.9, consistent differences in electroencephalographic (EEG) **sleep** measures were found. The subgroup of demented patients with a RBC/plasma choline ratio > 1.9 was more impaired on the Blessed Dementia Rating Scale and had less rapid eye movement (**REM**) **sleep** than the subgroup with a choline ratio ≤ 1.9. Similarly, depressives with a choline ratio ≤ 1.9 had a lower **REM** latency than depressives with a choline ratio > 1.9. Finally, depressed-demented (i.e., mixed-symptom) patients with a choline ratio > 1.9 showed less **sleep** continuity disturbance but more indeterminate non-**REM sleep** (reflecting loss of spindles and K-complexes) than those with lower choline ratios. These differences parallel those previously reported for **diagnostically** 'pure' depressed and demented patients, and they suggest a possible link between peripheral RBC/plasma choline measures and central nervous system function as reflected in **sleep** physiological alterations.

IT Major Concepts
Behavior; **Blood** and Lymphatics (Transport and Circulation); Geriatrics (Human Medicine, Medical Sciences); Metabolism; Neurology (Human Medicine, Medical Sciences); Pathology; Psychiatry (Human Medicine,. . .

IT Miscellaneous Descriptors
RAPID EYE MOVEMENT **SLEEP**

=> d his

(FILE 'HOME' ENTERED AT 11:27:30 ON 05 DEC 2005)

FILE 'STNGUIDE' ENTERED AT 11:27:35 ON 05 DEC 2005

FILE 'HOME' ENTERED AT 11:27:41 ON 05 DEC 2005

FILE 'BIOSIS' ENTERED AT 11:27:49 ON 05 DEC 2005
L1 6890 S (REM AND SLEEP)
L2 613 S L1 AND (BLOOD OR SERUM OR SERA)
L3 1806 S (DIAGNOS? OR PROGNOS? OR DETERMIN?) (3W) L1

```
L4          183 S L3 AND (BLOOD OR SERUM OR SERA)
L5            4 S L4 AND PROTEIN
L6            0 S L4 AND ELECTROPHORESIS
L7            8 S L4 AND MARKER

=> s l4 and (proteinase)
         27239 PROTEINASE
          9715 PROTEINASES
         31551 PROTEINASE
                 (PROTEINASE OR PROTEINASES)
L8            0 L4 AND (PROTEINASE)

=> s l4 and (glycosylated)
         18637 GLYCOSYLATED
L9            0 L4 AND (GLYCOSYLATED)

=> log y
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                      ENTRY     SESSION
FULL ESTIMATED COST                                   60.65       61.13

STN INTERNATIONAL LOGOFF AT 11:44:18 ON 05 DEC 2005
```